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Issue Date: 08 August 2007

Case Nos.: 2007-BLA-5079

In the Matter of:

H. P.,
Claimant

v.

Premium Energy, Inc.,
Employer

and

Director, Office of Workers' Compensation Programs,
Party-In-Interest

DECISION AND ORDER AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act of 1977, 30 U.S.C. Section 901 et seq. In accordance with the Act and the regulations issued thereunder, the case was referred by the Director, Office of Workers' Compensation Programs, for a formal hearing.

Benefits under the Act are awardable to miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of miners who were totally disabled at the time of their deaths (for claims filed prior to January 1, 1982), or to the survivors of miners whose deaths were caused by pneumoconiosis. Pneumoconiosis is a dust disease of the lungs arising from coal mine employment and is commonly known as "black lung."

A formal hearing was scheduled and held before the undersigned on March 14, 2007 in Abingdon, Virginia. At the hearing, I admitted Administrative Law Judge Exhibits (ALJX) 1 to 4, Director's Exhibits (DX) 1 to 32, Claimant's Exhibits (CX) 1 to 6, and Employer's Exhibits

(EX) 1 to 6. On April 30, 2007, I issued an Order admitting additional Employer's exhibits,[1] and closing the record. The parties were provided with time to submit written briefs. The Claimant submitted a brief on May 30, 2007; the Employer submitted a brief on June 4, 2007; the Director did not submit a brief.

I have based my analysis on the entire record, including the exhibits and representations of the parties, and have given consideration to the applicable statutory provisions, regulations, and case law, and made the following findings of fact and conclusions of law.

JURISDICTION AND PROCEDURAL HISTORY

The Claimant, H. P., filed his claim for benefits on December 16, 2005, which was granted by the District Director on July 28, 2006 (DX 2, 22). The WVCWP Fund requested a hearing, and on October 12, 2006, the claim was referred to the Office of Administrative Law Judges (DX 30).

ISSUES

The following issues are contested by the Employer.

1. Whether Mr. P. has pneumoconiosis.
2. If so, whether Mr. P.'s pneumoconiosis arose from his coal mine employment
3. Whether Mr. P. has a totally disabling respiratory impairment.
4. If so, whether his totally disabling respiratory impairment is due to pneumoconiosis.

(DX 30; Tr. 20-21).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Background

The Claimant, H. P., was born on May 19, 1939 (DX 2). He completed the seventh grade in school (DX 2). He married his wife, V. E. H., on December 27, 1950; they reside together (DX 2). Mr. P. has no children who are under 18 or dependent upon him. I find that Mr. P. has one dependent, namely his wife, for purposes of augmentation of benefits.

At the hearing, Mr. P. testified that he ran heavy equipment, most of the time a dozer, in a strip mine, for about 27 years (Tr. 22). Mr. P. was on oxygen, which had been prescribed by Dr. Grover in Kingsport (Tr. 23). Dr. Grover has treated Mr. P. since 2001. In addition to the oxygen, which Mr. P. has been on for about a year, Dr. Grover prescribes an inhaler and a breathing pill (Tr. 24). Mr. P. testified that he started smoking at about age 18, a pack a day until he quit thirty years ago (Tr. 25).

[1] This exhibit consists of interpretations by Dr. Paul Wheeler of a December 21, 2006 x-ray, a March 23, 2006 CT scan, and a February 7, 2003 CT scan. I have designated it as Employer's Exhibit 7.

The Director determined that Mr. P. has 27 years of coal mine employment. At the hearing, the Employer agreed that the Mr. P. worked for 27 years in coal mine employment; the Employer does not contest its status as the responsible operator (Tr. 21). This is amply supported by the evidence of record, including Mr. P.'s Social Security Earnings records, and his pay records (DX 6). Mr. P. stopped working in the coal mines on April 6, 2001, because he was disabled (DX 2). I find that Mr. P. has established 27 years of coal mine employment, and that the Employer is properly named as the responsible operator.

Medical Evidence

I have considered the following medical evidence under the limitations of the regulations.

X-ray Evidence

Exhibit No.	***Date of X-ray***	***Reading Date***	***Physician/ Qualifications***	***Impression***
CX 4	4-9-01	4-9-01	McReynolds	Multifocal areas of consolidation involving apices bilaterally, and right middle lobe; consistent with pneumonia in appropriate clinical setting
CX 6	5-4-01	5-4-01	Foster	Fibrotic scars in both apices likely related to confluent shadows of coal workers' pneumoconiosis with haziness at left lung base
CX 6	5-18-01	5-18-01	Foster	Substantial decrease in left pleural effusion, bilateral upper lobe chronic scarring consistent with pneumoconiosis
CX 6	7-12-01	7-12-01	Grover	Significant abnormalities, upper lung field greater than lower lung field nodular infiltrates, linear densities, pleural thickening in apices, consistent with severe coal workers' pneumoconiosis
CX 6	11-15-01	11-15-01	Grover	Extensive parenchymal scarring with upper lobe and apical predominance, pleural parenchymal changes suggestive of pneumoconiosis; extensive perihilar infiltrate scarring consistent with pneumoconiosis.
CX 6	3-21-02	3-21-02	Grover	Extensive parenchymal scarring in upper lungs bilaterally, with perihilar scarring and retractions

Exhibit No.	*Date of X-ray*	*Reading Date*	*Physician/ Qualifications*	*Impression*
CX 6	8-15-02	8-15-02	Lepsch	Diffuse reticular nodular pattern, with more extensive conglomerate opacities in upper lungs
CX 6	1-26-04	1-26-04	Lepsch	Chronic apical opacities suggesting pulmonary scarring; hila retracted upward
CX 6	5-12-05	5-12-05	Grover	Significant apical infiltrates in upper lung fields, right greater than left
CX 6	9-22-05	9-22-05	Grover	Extensive upper lobe scarring with retractions, some haziness at bases consistent with upper lobe retractions. Dense consolidation and scarring in perihilar areas and upper lung fields. Scarring so extensive that subtle parenchymal changes could not be ruled out.
DX 11	3-9-06	3-10-06	Rasmussen/B	1/1, r, r; Category A opacities
DX 12	3-9-06	3-29-06	Navani/B, BCR	Read for quality purposes
DX 13	3-9-06	5-23-06	Alexander/B, BCR	1/2, r, r; Category A opacities
EX 1	3-9-06	1-4-07	Wheeler/B, BCR	0/1, t, q
EX 2	3-9-06	1-4-07	Scott/B, BCR	Negative for pneumoconiosis
EX 2	3-9-06	1-4-07	Scatarige/B, BCR	Negative for pneumoconiosis
CX 4	8-21-06	8-21-06	Mullens	Interstitial nodularity with coalescent parenchymal scarring in upper lung zones consistent with silicosis; silicosis with progressive massive fibrosis, cardiomegaly, and previous CABG
EX 7	12-21-06	4-5-07	Wheeler/B, BCR	Negative for pneumoconiosis
CX 1	12-21-06	12-21-06	DePonte/B, BCR	1/2, q, r, category B opacities

Pulmonary Function Studies

Exhibit No.	*Date*	*Age/Ht*	*FEV1*	*FVC*	*MVV*	*Effort*
DX 11	3-9-06	66/73”	2.58 2.70*	3.83 3.74 8		Good
CX 6	8-3-06	67/74”	2.39	3.51		Good

CX 6	9-22-05	66/72”	2.67	3.79		Good
CX 6	2-12-04	64/73”	2.90	4.24		Good
CX 6	3-21-02	62/73”	2.78	4.04		
CX 6	8-23-01	62/73”	2.05	3.01		

* Results after administration of bronchodilator

Arterial Blood Gas Studies

Exhibit No.	***Date***	***Physician***	***pCO2***	***pO2***	***At rest/exercise***
DX 11	3-9-06	Rasmussen	32	78	At rest

CT Scans

The records from Pulmonary Associates include the results of a chest CT scan performed on March 23, 2006 (CX 6). Dr. John M. McMurray read the CT scan, noting numerous calcified lymph nodes in the mediastinum and hilar areas, many of which had a classic “eggshell” appearance. There were also numerous irregular opacities, most prominent in the mid and upper lobes, as well as several conglomerate opacities in both lungs, which were probably areas of progressive mass fibrosis associated with pneumoconiosis. Dr. McMurray noted an area about 5 cm by 3.1 cm in the right upper lobe, one about 5.8 cm by 4.3 cm with irregular margins in the right mid lung, and a conglomerate density in the left upper lobe about 3.9 cm in maximum diameter. There were numerous other small reticular and nodular opacities, as well as nonspecific areas of pleural thickening. He felt that the pattern was consistent with coal workers’ pneumoconiosis. He noted that the eggshell type calcifications involving the lymph nodes can also be seen in silicosis. Dr. McMurray suspected areas of progressive massive fibrosis in both lungs. Dr. McMurray’s conclusion was that the findings were consistent with coal workers’ pneumoconiosis and/or silicosis. He saw extensive progressive massive fibrosis in both lungs, most prominent in the upper lobes, and extensive calcified lymph nodes.

Dr. Wheeler reviewed the March 23, 2006 CT scan (EX 7). In his opinion, it showed advanced calcified granulomatous disease, most likely histoplasmosis, with masses in the lung involving the pleura and calcified granulomata in the hila and mediastinal nodes.

Mr. P. underwent a chest CT scan on February 7, 2003 at the Holston Valley Medical Center, which was reviewed by Dr. Thomas F. Pugh (CX 5). He noted fibronodular opacities throughout both lungs, predominantly in an upper lobe distribution. There were confluent areas of scarring and pleural parenchymal thickening in both upper lobes and suprahilar regions. There were numerous mediastinal lymph nodes with peripheral calcification, and coronary arterial calcifications. He favored silicosis as the etiology, although he noted that sarcoidosis and other pneumoconiosis were also diagnostic considerations.

Dr. Wheeler also reviewed the February 7, 2003 CT scan, noting irregular masses in the posterior upper lobes and apices, and superior segment of the right lower lung (EX 7). He also noted a small mass in the anterior right upper lung, all involving the pleura, compatible with conglomerate granulomatous disease, histoplasmosis more likely than tuberculosis, with adjacent linear and irregular fibrosis indicating at least some healing. Dr. Wheeler described calcified granulomata in the bilateral hilar and peritracheal nodes and in the subcarinal nodes, due to healed histoplasmosis more likely than tuberculosis. There were a few small nodules in the posterior lower lobes mixed with linear scars, compatible with histoplasmosis, some involving the pleura. He also noted minimal smooth right lower posterolateral pleural fibrosis from healed inflammatory disease. Dr. Wheeler noted no symmetrical small nodular infiltrates in the mid and upper lungs which could indicate pneumoconiosis, but stated that an exact diagnosis was needed for any significant disease to assure proper therapy, and that it should have been made easily with biopsy.

Mr. P. underwent a chest CT scan on August 15, 2002, which was read by Dr. Thomas C. Lepsch (CX 5). Dr. Lepsch noted diffuse lung disease with a central and upper lung predominance, consisting of peribronchovascular thickening, and small nodules. There were also larger nodules and mass-like areas associated with fibrosis, and limited subpleural honeycombing. He noted limited mediastinum hilar lymph node enlargement and calcification. He felt that the findings suggested sarcoidosis, although inhalational disease such as silicosis or coal miners' pneumoconiosis, were also possible. He described a diffuse reticular nodular pattern in the lungs, with more extensive conglomerate opacities in the upper lungs.

Dr. Thomas Pugh read the CT scan performed on August 15, 2002 (CX 5). He noted fibronodular opacities distributed throughout both lungs, predominantly in an upper lobe distribution. There were confluent areas of scarring and pleural parenchymal thickening in both upper lobes and suprahilar regions; mediastinal images showed numerous mediastinal lymph nodes with peripheral calcification. Dr. Pugh's favored etiology was silicosis, although sarcoidosis and other pneumoconioses were also diagnostic considerations.

Dr. John Siner reviewed a CT scan performed on April 20, 2001 (CX 6). He noted a small left pleural effusion, and patchy alveolar infiltrates in the upper lobes of both lungs, associated with some calcification. There were calcified lymph nodes in the mediastinum. His impression was extensive bilateral pulmonary emboli, with bilateral upper lobe alveolar infiltrates suggestive of pneumonia or progressive massive fibrosis.

Medical Opinion Evidence

Dr. D. L. Rasmussen

Dr. Rasmussen examined Mr. P. on March 9, 2006 at the Director's request (DX 11). He reported Mr. P.'s employment history, as well as his medical and family histories, and his symptoms. Mr. P. told Dr. Rasmussen that he smoked a half pack of cigarettes from 1956 to 1986. On his examination of Mr. P., Dr. Rasmussen noted moderately reduced breath sounds on auscultation, with widespread rhonchi, and prolonged expiratory phase with forced expirations.

Mr. P.'s x-ray showed pneumoconiosis 1/1, r, r, with category A opacities. His pulmonary function and arterial blood gas study results were normal.

Dr. Rasmussen concluded that Mr. P. has complicated pneumoconiosis, category A; chronic bronchitis with a productive cough; ASHD, with a myocardial infarction in 2001 and 2003; and atrial fibrillation. He attributed Mr. P.'s complicated pneumoconiosis as well as his chronic bronchitis to his exposure to coal mine dust. Dr. Rasmussen felt that Mr. P.'s ASHD and atrial fibrillation were due to a non occupational factor. According to Dr. Rasmussen, Mr. P. has minimal loss of lung function, as reflected by his ventilatory impairment and reduction in single breath diffusing capacity. Based on the resting study results, Mr. P. retained the pulmonary capacity to perform his last regular coal mine job.

He noted that Mr. P. had a significant history of exposure to occupational dusts, including 27 years of coal mine employment. He had radiographic changes consistent with complicated pneumoconiosis, category A. Dr. Rasmussen felt that it was medically reasonable to conclude that Mr. P. had complicated pneumoconiosis, that arose as a consequence at least in part of his coal mine dust exposure. According to Dr. Rasmussen the two causes of Mr. P.'s minimal impairment were his cigarette smoking and his coal mine dust exposure, with his coal mine dust exposure being a significant contributing factor. Mr. P. had clinical complicated coal workers' pneumoconiosis, which contributed significantly to his minimal impairment.

Dr. Samuel V. Spagnolo

Dr. Spagnolo reviewed Mr. P.'s medical records at the Employer's request, and prepared a report dated January 27, 2007 (EX 3). He stated that it appeared that Mr. P. worked for 27 years in surface coal mining employment, which would have placed him at risk for developing pneumoconiosis. According to Dr. Spagnolo, there was conflicting information about Mr. P.'s smoking history, with a number of examiners reporting a much longer and more intense smoking history than indicated by Mr. P. in his answers to interrogatories.

Dr. Spagnolo reported that Mr. P. had suffered two myocardial infarctions, coronary artery bypass surgery, and coronary angioplasty with stent placement. He also developed congestive heart failure and renal failure. Mr. P.'s medical history included deep venous thrombosis, pulmonary emboli, hyperlipidemia, and diabetes mellitus. He has complained of intermittent wheezing, cough, chest pain, and shortness of breath. Dr. Spagnolo indicated that physical examinations showed findings most consistent with congestive heart failure.

Dr. Spagnolo noted that Mr. P.'s spirometry values were normal in 2002, 2004, and 2005, and his arterial blood gas values were within normal limits in 2006. He indicated that Dr. Rasmussen noted only a minimal obstructive ventilatory impairment, at a time when it appeared that Mr. P. was in congestive heart failure. According to Dr. Spagnolo, Mr. P.'s chest x-rays showed consistent evidence for the presence of parenchymal abnormalities consistent with both granulomatous disease and pneumoconiosis. He placed great weight on Dr. Wheeler's report on the March 2006 x-ray, noting that he was a pre-eminent radiologist in the evaluation of x-rays of persons with occupational exposure and related lung disease. Dr. Wheeler concluded that the x-

ray changes were most consistent with the effects of a chronic granulomatous process, not pneumoconiosis.

Dr. Spagnolo stated that respiratory symptoms and breathing difficulties are frequently an early manifestation of underlying cardiac disease; impaired left heart function can result in exercise intolerance, manifested as exertional dyspnea and fatigue. These are frequently the primary symptoms of systolic or diastolic heart failure. According to Dr. Spagnolo, other symptoms and signs of left heart failure can include orthopnea, paroxysmal nocturnal dyspnea, early morning wheezing or intermittent wheezing, cough, reduced DLCO value, variable airflow obstruction, and variability in arterial p02. In his opinion, Mr. P.'s cardiac disease was responsible for his respiratory complaints.

Dr. Spagnolo concluded that Mr. P. does not have a pulmonary/respiratory impairment or condition that has been aggravated in any way by his inhalation of coal mine dust. Based on the results of Mr. P.'s recent tests, Dr. Spagnolo felt that he retained the respiratory capacity to perform his previous coal mine employment as a heavy equipment operator; this position would not change even if it were determined that Mr. P. had pneumoconiosis.

Dr. Spagnolo prepared a supplemental report dated February 11, 2007, after reviewing x-ray interpretations by Dr. Scott and Dr. Scatarige of the March 9, 2006 x-ray (EX 4). He stated that their reports provided additional strong evidence that the x-ray changes noted in Mr. P. do not represent pneumoconiosis.

Dr. Lawrence J. Repsher

Dr. Repsher reviewed Mr. P.'s records at the Employer's request, and prepared a report dated February 7, 2007 (EX 5). He concluded that Mr. P. did not suffer from either medical or legal coal workers' pneumoconiosis or any other pulmonary or respiratory disease or condition either caused by or aggravated by his inhalation of coal mine dust. He noted that there was no radiographic evidence of pneumoconiosis. He acknowledged that Mr. P.'s x-ray and CT scan were quite abnormal, but they were most consistent with tuberculosis or sarcoidosis, and not consistent with pneumoconiosis. According to Dr. Repsher, the only reliable board-certified radiologist and B readers found no evidence of complicated or simple pneumoconiosis.

According to Dr. Repsher, there was no histologic evidence of pneumoconiosis, and no pulmonary function test evidence of pneumoconiosis. He noted that the only available pulmonary function study results available showed only mild and clinically insignificant COPD, which he felt was overwhelmingly most likely due to Mr. P.'s long cigarette smoking habit. Dr. Spagnolo noted that Mr. P.'s arterial blood gas study results were within normal limits for his age and altitude; thus, there was no arterial blood gas evidence of pneumoconiosis.

Because Mr. P. had no clinically significant pulmonary impairment, Dr. Repsher felt that clearly from a respiratory point of view, he was fully fit to perform his usual coal mine work or work of a similar arduous nature. But Mr. P. suffers from a number of other serious and potentially serious diseases and condition, none of which could be fairly attributed to his

exposure to coal mine dust. They are diseases and conditions of the general population, primarily related to heredity and lifestyle factors.

Dr. Paul S. Wheeler

Dr. Wheeler testified by deposition on February 28, 2007 (EX 6). He described granulomatous disease as an infection such as tuberculosis or histoplasmosis, or a noninfectious process such as sarcoid. According to Dr. Wheeler, histoplasmosis is a fungal infection that can cause significant respiratory impairment. He stated that it leaves scarring, and masses up to 8 centimeters in diameter, involving the lungs, pleura, and lymph nodes. Dr. Wheeler described the pleura and the parenchyma, indicating that the pleura is simply a protective lining, so that when disease involves the pleura, it typically is more likely to be inflammatory, such as granulomatous disease or possibly cancer.

According to Dr. Wheeler, tuberculosis is one of the very few diseases that attacks the apices; histoplasmosis and emphysema do also, but silicosis and coal workers' pneumoconiosis typically do not involve the apices or periphery of the lungs; they certainly do not involve the pleura, which has no alveoli. But they can involve the apices and periphera if there is extensive central mid and upper lung involvement, and it has a spill-over phenomenon. In Mr. P.'s case, the disease is primarily in the apices, which is out of the zone for silicosis or coal workers' pneumoconiosis.

Referring to his statement that there was unknown activity at least partly healed, Dr. Wheeler stated that he thought that there was fibrosis, at least in the apex and involving the pleura. There were also calcified granulomata, indicating that the disease process was healed.

Dr. Wheeler testified that in Mr. P.'s case, he favored histoplasmosis over tuberculosis; in his experience, calcified granulomata are far more frequently seen in cases of healed histoplasmosis than tuberculosis. Both can cause calcified granulomata, but histoplasmosis is much more common, and much more likely to self-cure without therapy. Dr. Wheeler stated: "So my feeling is if a person has a negative tuberculin test and mass lesions in the lungs and the mass lesions are known not to be cancer than [sic] the most likely diagnosis is histoplasmosis."

According to Dr. Wheeler, the vast majority of pneumoconioses give symmetrical patterns, unless the lungs are distorted either by partial resection from a previous operation, or emphysema with bullous blebs that distort the lungs. With silicosis and coal workers' pneumoconiosis, the inhaled dust is typically deposited in the central portion of the mid and upper lungs. It is a symmetrical central nodular pattern in the mid and upper lungs. Noting that he had considered a diagnosis of pneumoconiosis, Dr. Wheeler stated that if the nodules he saw had been primarily round and small, and symmetrical and in the central portion of the mid and upper lungs, he would have given a much higher classification. But they were not, and there were porous irregular opacities in addition to the nodules. He indicated that he would be better able to do staging with a CT scan, which are acceptable in the medical community to make diagnoses.

In Dr. Wheeler's opinion, Mr. P. has granulomatous disease with mass lesions, or conglomerate granulomatous disease. He felt that it was possible that some of the nodules were from coal workers' pneumoconiosis, but they would have to be in the central portion of the mid and upper lungs, not in the apices or involving the pleura. He stated that he would like to see a biopsy, which should have been done in Mr. P.'s case.

Dr. Wheeler testified that complicated pneumoconiosis is quite rare, and was seen most frequently in drillers working unprotected during and before World War II, when there was no requirement for miners to wear respiratory protection.

According to Dr. Wheeler, it is not possible to make an exact diagnosis of a mass on a chest x-ray. In order to make a diagnosis, a biopsy is necessary. He noted that Mr. P. had a four by five centimeter mass in the lateral right upper lobes involving the pleura, out of the strike zone for a typical large opacity of coal workers' pneumoconiosis.

Richlands Community Medical Center

The exhibit file includes treatment notes from the Richlands Community Medical Center (CX 3). These records are handwritten, and almost completely illegible. They include three handwritten x-ray reports; the date of the report is illegible, and there is no signature, only initials. As far as can be deciphered, they appear to report a mass in Mr. P.'s lungs due to conglomerate pneumoconiosis, and marked COPD due to complicated pneumoconiosis.

Johnston Memorial Hospital

The exhibit file includes records from the Johnston Memorial Hospital, where Mr. P. was admitted on August 22, 2006 with complaints of chest tightness and shortness of breath (CX 4). Dr. Wiley Kent, who prepared the discharge summary, reported discharge diagnoses of cor pulmonale, congestive heart failure, atrial fibrillation, silicosis with progressive massive fibrosis, diabetes, hypertension, hyperlipidemia, cholelithiasis, renal insufficiency, pulmonary hypertension, chronic obstructive pulmonary disease, coronary artery disease, and coal workers' pneumoconiosis. Dr. Kent noted that Mr. P. had a history of COPD and coal workers' pneumoconiosis. According to Dr. Kent, Mr. K. had cor pulmonale due to his right ventricular hypertrophy on EKG, increased venous pressure, and edema, and it was suspected that he had severe pulmonary hypertension due to his progressive massive fibrosis from the silicosis disease.

Dr. Larry Cox saw Mr. P. in consultation. He noted that Mr. P.'s chest x-ray on admission showed progressive massive fibrosis. After examining Mr. P., Dr. Cox concluded that he had abdominal bloating and discomfort, which he suspected was due to passive congestion from his right heart failure; gallbladder disease was also a possibility. He also felt that Mr. P. had cor pulmonale, with right ventricular hypertrophy on EKG, and increased venous pressure and edema. Dr. Cox suspected that Mr. P. had severe pulmonary hypertension related to progressive massive fibrosis. He noted atrial fibrillation with slow ventricular response. Dr. Cox also diagnosed silicosis with progressive massive fibrosis; status post coronary bypass surgery; diabetes; hypertension; dyslipidemia; gallstones; and moderately severe renal dysfunction.

Holston Valley Medical Center

Mr. P. went to the Holston Valley Medical Center emergency room on January 26, 2004 with complaints of chest pain (CX 5). Dr. Kathy Burniston, who completed his history and physical, noted a medical history including extensive bilateral pulmonary emboli and DVT in May 2001, diabetes mellitus type II, chronic obstructive pulmonary disease/coalworkers' pneumoconiosis with extensive pulmonary fibrosis, hyperlipidemia, and hypertension. Mr. P. underwent left heart catheterization and grafting. Her diagnoses on Mr. P.'s discharge on January 29, 2004 were non-ST elevation MI, chronic obstructive pulmonary disease, hypertension, dyslipidemia, diabetes, and history of atrial fibrillation.

Mr. P. was admitted on April 18, 2001 for chest pain. Dr. Bruce Grover noted that he had a history of silicosis and coal workers' pneumoconiosis and COPD, as well as hypercholesterolemia and hypertension. He suspected that Mr. P. had increased symptoms related to chronic underlying pulmonary fibrosis and some degree of COPD, as well as some persistent edema. Dr. Grover's diagnoses on Mr. P.'s discharge on April 27, 2001 included respiratory distress with hypoxemia, pulmonary embolus, pulmonary fibrosis secondary to progressive massive fibrosis, silicosis/pneumoconiosis, exercise intolerance, coronary disease status post coronary bypass surgery, anemia, and elevated ANA. Dr. Shelly Hearn also prepared a list of discharge diagnoses, which included bilateral pulmonary emboli and right calf deep venous thrombosis, dyspnea, pulmonary fibrosis, coronary artery disease, congestive heart failure, hypercholesterolemia, hypertension, and chronic obstructive pulmonary disease.

Mr. P. went to the hospital on April 9, 2001 and was discharged on April 15, 2001. He underwent cardiac catheterization and emergent coronary revascularization, and repair of an impending rupture. Dr. Richard Feit prepared the discharge summary, with diagnoses of coronary artery disease, status post emergent CABG times three, and repair of impending ventricular rupture; status post recent myocardial infarction; hypercholesterolemia; hypertension; and remote history of tobacco abuse.

Pulmonary Associates of Kingsport

The record includes treatment notes from Pulmonary Associates of Kingsport, where Mr. P. was treated by Dr. Bruce Grover and Dr. Robert Rosser (CX 6). Mr. P. was treated for shortness of breath, dyspnea on exertion, coal workers' pneumoconiosis with progressive massive fibrosis, asymmetric lower extremity edema. In a treatment noted dated February 12, 2004, Dr. Rosser noted that a pulmonary function study showed no significant obstructive or restrictive impairment; lung volumes are normal, and there was a borderline decrease in the DLCO. In a treatment note dated February 13, 2003, Dr. Grover noted that a CT scan showed stable fibronodular opacities in both lungs with confluent scarring; his impression was coal workers' pneumoconiosis with extensive scarring and mass-like opacities that were stable by CT scan; COPD based on chronic obstructive disease; and restrictive lung disease.

In a treatment note dated March 21, 2002, Dr. Grover reported that an x-ray showed extensive scarring in the upper lung field. Pulmonary function studies showed improved

combined obstructive and restrictive disease. Dr. Grover's impression was coal workers' pneumoconiosis with extensive scarring; COPD; head cold; history of PE. In a note dated July 12, 2001, Dr. Grover reported that Mr. P.'s x-ray showed significant parenchymal scarring consistent with coal workers' pneumoconiosis, although he could not rule out other etiologies.

Dr. Foster completed a treatment note dated May 18, 2001, noting that x-rays showed bilateral upper lobe chronic scarring consistent with pneumoconiosis. He reported on a May 4, 2001 note that x-rays showed fibrotic type scars in both lung apices, likely related to confluent shadows of coal workers' pneumoconiosis.

Randy's Gateway Drug

Mr. P. submitted records from Randy's Gateway Drug reflecting his pharmacy purchase from 2003 through 2006 (CX 2).

DISCUSSION

Existence of Pneumoconiosis

Pneumoconiosis is defined, by regulation, as a "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 20 C.F.R. § 718.201. The regulations at 20 C.F.R. § 718.203(b) provide that, if it is determined that the miner suffered from pneumoconiosis and has engaged in coal mine employment for ten years or more, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. If, however, it is established that the miner suffered from pneumoconiosis but worked less than ten years in the coal mines, then the claimant must establish causation by competent evidence. *Stark v. Director, OWCP*,9 B.L.R. 1-36 (1986); *Hucker v. Consolidation Coal Co.*,9 B.L.R. 1-137 (1986). The claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See, Director, OWCP v. Greenwich Collieries,* 114 S.Ct. 2251 (1995).

Because the current claim was filed after the enactment of the Part 718 regulations, the evidence will be evaluated under standards found in 20 C.F.R. Part 718. The existence of pneumoconiosis may be established by any one or more of the following methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a). I have independently assessed the evidence under each of these methods.

To establish the existence of pneumoconiosis, a chest x-ray must be classified as category 1, 2, 3, A, B, or C, according to the ILO-U/C classification system. A chest x-ray classified as category 0, including subcategories 0/1, 0/0, or 0/-, does not constitute evidence of pneumoconiosis. In this case, the record includes seven ILO interpretations of two x-rays, both performed in 2006. The first, performed on March 9, 2006, was interpreted as positive by Dr. Rasmussen, who is a B reader, and Dr. Alexander, who is dually qualified, but as negative by Dr. Scott, Dr. Wheeler, and Dr. Scatarige, who are dually qualified. Given the preponderance of

negative readings by the most highly qualified interpreters, I find that this x-ray is not positive for pneumoconiosis.

The second x-ray, done on December 21, 2006, was read as positive by Dr. DePonte, who is dually qualified, and as negative by Dr. Wheeler. I find that these interpretations are at best in equipoise, and thus this x-ray is not positive for pneumoconiosis.

In addition to the ILO interpretations, there are also a number of narrative interpretations by Mr. P.'s treating physicians, covering the time period from 2001 through 2006. These reports describe scarring and densities, many indicating that the findings are consistent with pneumoconiosis. The most recent, by Dr. Mullens, reports silicosis with progressive massive fibrosis. However, the qualifications of the interpreting physicians are not in the record, and while the findings certainly support the interpretations by Dr. Rasmussen and Dr. Alexander, I find that, in view of the qualifications of Dr. Wheeler, Dr. Scott, and Dr. Scatarige, they are not sufficient to tip the balance in favor of a finding of pneumoconiosis

There is no autopsy or biopsy evidence in the record, nor, with the exception of Section 718.304, discussed further below, do any of the statutory presumptions apply.

Under 20 C.F.R. §718.304, there is an irrebuttable presumption that a miner is totally disabled due to pneumoconiosis if the miner is suffering from "complicated pneumoconiosis," although that term does not appear in the statute. A miner can establish complicated pneumoconiosis if he suffers from a chronic dust disease of the lung which:

> (a) When diagnosed by chest x-ray … yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C…; ***or***
>
> (b) When diagnosed by biopsy or autopsy, yields massive lesions in the lung; ***or***
>
> (c) When diagnosed by means other than those specified in paragraphs (a) and (b) of this section, would be a condition which could reasonably be expected to yield the results described in paragraph (a) or (b) of this section had diagnosis been made as therein described: Provided, however, That any diagnosis made under this paragraph shall accord with acceptable medical procedures.

20 C.F.R. §718.304 (emphasis added); s*ee Eastern Associated Coal Corp. v. Director, OWCP*, 220 F.3d 250 (4th Cir. 2000). The Fourth Circuit has recently described the appropriate analysis under Section 21(c)(3) of the Act and the implementing regulations at 20 C.F.R. §718.304:[2]

[2] It is important to note that Section 21(c)(3) of the Act, 30 U.S.C. §921(c)(3), and Section 718.304 of the implementing regulations, 20 C.F.R. §718.304, are virtually identical in language, and the Fourth Circuit has treated them as interchangeable for purposes of invoking the irrebuttable presumption. *See Eastern*, 220 F.3d 250.

> While 30 U.S.C. §921(c)(3) sets forth, in clauses (A), (B), and (C), three different ways to establish the existence of statutory complicated pneumoconiosis for purposes of invoking the irrebuttable presumption, these clauses are intended to describe a single, objective condition. . . And, because prong (A) sets out an entirely objective scientific standard—i.e. an opacity on an x-ray greater than one centimeter—x-ray evidence provides the benchmark for determining what under prong (B) is a massive lesion and what under prong (C) is an equivalent diagnostic result reached by other means.
>
> Prongs (A), (B), and (C) are stated in the disjunctive; therefore a finding of statutory complicated pneumoconiosis may be based on evidence presented under a single prong. But the ALJ must in every case review the evidence under each prong of §921(c)(3) for which relevant evidence is presented to determine whether complicated pneumoconiosis is present. Evidence under one prong can diminish the probative force of evidence under another prong if the two forms of evidence conflict. Yet, a single piece of relevant evidence can support an ALJ's finding that the irrebuttable presumption was successfully invoked if that piece of evidence outweighs conflicting evidence in the record. Thus, even where some x-ray evidence indicates opacities that would satisfy the requirements of prong (A), if other x-ray evidence is available or if evidence is available that is relevant to an analysis under prong (B) or (C), then all of the evidence must be considered and evaluated to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray. Of course, if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force only if other evidence affirmatively shows that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problems with the equipment used, or incompetence of the reader.

Eastern, 220 F.3d at 255-6 (internal quotations and citations omitted). Furthermore, the Fourth Circuit emphasized that the parties should not assume "that the statutory definition of 'complicated pneumoconiosis' must be congruent with a medical or pathological definition." *Id.* at 257. Instead, it is important to remember in the determination of complicated pneumoconiosis that the presumption under 20 C.F.R. §718.304 "is triggered by a congressionally defined condition." *Id.* In other words, invocation of the irrebuttable presumption does not require any additional clinical finding if prong (A), (B), or (C) is met.

The Court noted that the statute creating the irrebuttable presumption of causation does not refer to the condition as "complicated pneumoconiosis," or to a medical condition that doctors have independently called complicated pneumoconiosis. As the Court stated:

> [T]he presumption under § 921(c)(3) is triggered by a congressionally defined condition, for which the statute gives no name but which, if found to be present, creates an irrebuttable presumption that disability or death was caused by pneumoconiosis. . . . In short, the statute betrays no intent to incorporate a purely medical definition.

Eastern Associated Coal Corporation, 250 F.3d at 257.

Thus, if Mr. P. meets the congressionally defined condition, that is, if he establishes that he has a condition that manifests itself on x-rays with opacities greater than one centimeter, he is entitled to the irrebuttable presumption of total disability due to pneumoconiosis, unless there is affirmative evidence under prong A, B, or C that establishes either that these opacities do not exist, or that they are the result of a disease process unrelated to his exposure to coal mine dust.

In this case, the record includes three interpretations with findings of category A or B opacities. Dr. Rasmussen and Dr. Alexander both reported category A opacities on Mr. P.'s March 9, 2006 x-ray, while Dr. Wheeler, Dr. Scott, and Dr. Scatarige found these x-rays to be negative for pneumoconiosis. With respect to Mr. P.'s December 21, 2006 x-ray, Dr. DePonte found category B opacities, while Dr. Wheeler interpreted this x-ray as negative for pneumoconiosis. Thus, while there are x-ray interpretations that clearly satisfy the requirements of prong (A), there are other x-ray interpretations to the contrary, and I must consider all of them to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray. *Scarbro*, 220 F.3d at 255-56.

On his review of the March 9, 2006 x-ray, Dr. Wheeler described two masses, 5 X 4 cm., and 2-3 cm., which he felt were compatible with conglomerate granulomatous disease. Dr. Scott described infiltrates and/or fibrosis in the upper lungs extending to the pleura, probably due to tuberculosis or unknown activity. Dr. Scatarige noted a 5 cm. mass or confluent infiltrate in the right upper lungs, for which he favored tuberculosis.

On his review of the December 21, 2006 x-ray, Dr. Wheeler described a 7 cm. mass in the right upper lung, and a smaller irregular mass in the left apex and subapical left upper lung, which he felt were compatible with conglomerate granulomatous disease, probably histoplasmosis more likely than tuberculosis.

Based on the totality of the x-ray evidence, I find that Mr. P. has established that he has a process that shows up on his x-ray as category A or B opacities. Thus, Dr. Wheeler, Dr. Scott, and Dr. Scatarige acknowledge the presence of large masses on Mr. P.'s chest x-rays; their reports are not affirmative evidence that establishes that the large opacities identified by Dr. Rasmussen, Dr. Alexander, and Dr. DePonte are not there.

Additionally, the narrative x-ray reports in the record, while not sufficient to establish the presence of category A or B opacities, lend credibility to the findings of large opacities by Dr. Rasmussen, Dr. Alexander, and Dr. DePonte. Thus, interpretations from as early as 2001 reflect multifocal areas of consolidation (Dr. McReynolds, April 9, 2001); fibrotic scars in both apices (Dr. Foster, May 4, 2001); bilateral upper lobe chronic scarring (Dr. Foster, May 18, 2001); extensive parenchymal scarring (Dr. Grover, November 15, 2001, March 21, 2002); diffuse reticular nodular pattern with more extensive conglomerate opacities in upper lungs (Dr. Lepsch, August 15, 2002); chronic apical opacities suggesting pulmonary scarring (Dr. Lepsch, January 26, 2004); significant apical infiltrates in upper lung fields (Dr. Grover, May 12, 2005); and extensive upper lobe scarring with retractions at bases, dense consolidation and scarring in perihilar areas and upper lungs (Dr. Grover, September 22, 2005).

The record also includes evidence that falls under Prong (C), in the form of numerous CT scans. These reports date back to April 2001, when Dr. Siner reported extensive bilateral pulmonary emboli, with bilateral upper lobe alveolar infiltrates suggestive of pneumonia or progressive massive fibrosis. A little more than a year later, Dr. Pugh reported that Mr. P.'s August 2002 CT scan showed fibronodular opacities throughout both lungs, predominantly in the upper lobes, with confluent areas of scarring and pleural parenchymal thickening. He felt that these findings were due to silicosis, although sarcoidosis and other pneumoconioses were also possibilities. Dr. Lepsch also reviewed this CT scan, noting diffuse peribronchovascular thickening and small nodules predominantly in the central and upper lung, with larger nodules and mass-like areas associated with fibrosis. Dr. Lepsch felt that these findings suggested sarcoidosis, although silicosis or coal miner's pneumoconiosis were possibilities.

Dr. Pugh reviewed Mr. P.'s CT scan done the following year in February, noting fibronodular opacities throughout both lungs, predominantly in an upper lobe distribution, with confluent areas of scarring and pleural parenchymal thickening in both upper lobes and suprahilar regions. He felt that these findings were the result of silicosis, although sarcoidosis or other pneumoconioses were considerations. Dr. Wheeler also reviewed this CT scan, and described irregular masses in the upper lobes and apices, and right lower lung, as well as a small mass in the right upper lung, which he felt were compatible with conglomerate granulomatous disease. He also reported calcified granulomata in the hilar, peritracheal, and subcarinal nodes, due to healed histoplasmosis more likely than tuberculosis. But Dr. Wheeler did not find any symmetrical small nodular infiltrates in the mid and upper lungs to indicate pneumoconiosis.

Mr. P.'s most recent CT scan, on March 23, 2006, was reviewed by Dr. McMurray, who described numerous calcified lymph nodes in the mediastinum and hilar areas, many with a classic "eggshell" appearance, as well as numerous irregular opacities most prominent in the mid and upper lobes. He also described several conglomerate opacities in both lungs, which he felt were probably areas of progressive mass fibrosis associated with pneumoconiosis. In addition, there were numerous small reticular and nodular opacities, and nonspecific areas of pleural thickening. Dr. McMurray felt that the pattern was consistent with coal workers' pneumoconiosis, and noted that the eggshell type calcifications involving the lymph nodes can also be seen in silicosis. He suspected that there were areas of progressive massive fibrosis in both lungs, most prominent in the upper lobes.

Dr. Wheeler also reviewed this CT scan, concluding that it showed advanced calcified granulomatous disease, with masses involving the pleura, and calcified granulomata in the hila and mediastinal nodes.

While none of these CT scan interpretations state that the conglomerate masses would appear on an x-ray as an opacity of at least one centimeter in diameter, which is the standard set out by the Fourth Circuit in *Scarbro*, all of these interpretations lend credibility to the conclusion that Mr. P. has a process in his lungs that shows up on x-ray as an opacity of at least one centimeter in diameter, as reported by Dr. Rasmussen, Dr. DePonte, and Dr. Alexander. These reports certainly do not refute such a conclusion, and thus they are not affirmative evidence to establish that the opacities noted by Dr. Rasmussen, Dr. DePonte, and Dr. Alexander are not there.

Dr. Wheeler, who read Mr. P.'s x-rays and CT scans, conceded that it was "possible" that some of the nodules seen on x-ray and CT scan were the result of coal workers' pneumoconiosis, if they were in the central portion of the mid and upper lungs. He was not willing to concede this point without biopsy evidence, stating that this should have been done in Mr. P.'s case. Dr. Wheeler stated that he had considered a diagnosis of pneumoconiosis, but the nodules he saw were not primarily round, small, symmetrical, and in the central portion of the mid and upper lungs, and there were also porous irregular opacities. Dr. Wheeler stated that he would be better able to do "staging" with a CT scan. In fact, Dr. Wheeler reviewed two of Mr. P.'s CT scans, but he did not discuss their significance or even his own findings on review of those CT scans.

Dr. Rasmussen, Dr. DePonte, and Dr. Alexander concluded that the large opacities they designated on their ILO form were due to pneumoconiosis. This is supported by Mr. P.'s medical records, including the x-rays and CT scans reviewed by his treating physicians.[3] I find that the Employer has not offered affirmative evidence sufficient to establish that these large opacities are due to a process other than pneumoconiosis.

Dr. Wheeler stated that silicosis and coal workers' pneumoconiosis typically do not involve the apices or periphery; in Mr. P.'s case, his "disease" was primarily in the apices, out of the "zone" for silicosis or coal workers' pneumoconiosis. But the CT scan reports show that the fibronodular opacities, confluent areas of scarring, and thickening, while they may have "involved" the pleura and apices, were not confined to the apices or periphery. Indeed, in reviewing the most recent CT scan, Dr. McMurray described numerous irregular opacities most prominent in the mid and upper lobes, and several conglomerate opacities in both lungs. Dr. Pugh found fibronodular opacities throughout both lungs, predominantly in an upper lobe distribution, with confluent areas of scarring and pleural parenchymal thickening in both upper lobes and suprahilar regions. Dr. Wheeler himself described irregular masses in the posterior upper lobes and apices, and right lower lung, and a small mass in the right upper lung, all "involving" the pleura. Dr. Lepsch found diffuse peribronchovascular thickening and small nodules with a central and upper lung predominance, and larger nodules and mass-like areas

[3] While some of the x-ray and CT scan interpretations suggest other diagnostic possibilities, I find that the preponderance of those interpretations support the conclusion that the abnormalities in Mr. P.'s lungs are due to pneumoconiosis.

associated with fibrosis. Dr. Pugh also found fibronodular opacities throughout both lungs, predominantly in an upper lobe distribution, with confluent areas of scarring and pleural parenchymal thickening in both upper lobes and suprahilar regions. Dr. Siner, who reviewed the earliest CT scan, performed in 2001, noted infiltrates in the upper lobes of both lungs, with some calcification. These CT scan reports are consistent in that they all describe a process in Mr. P.'s upper lungs, consisting of fibronodular opacities with conglomerate masses.

Nevertheless, although he admitted the possibility that some nodules were from pneumoconiosis, and he did not have the biopsy evidence he felt should be available, or the CT scans for "staging," Dr. Wheeler concluded that Mr. P. has conglomerate granulomatous disease. But he did not explain why a finding of granulomatous disease necessarily precluded a finding of pneumoconiosis.

Finally, I find that the medical opinion evidence supports the conclusion that Mr. P. has established that he has both simple and complicated pneumoconiosis. Dr. Rasmussen, who examined Mr. P. at the Director's request, concluded that he had complicated pneumoconiosis based on his x-ray results, and chronic bronchitis due to his exposure to coal mine dust, as well as his cigarette smoking. His report is based on his clinical, x-ray, and test findings, and is consistent with Mr. P.'s past medical history, as reported by his treating physicians, and I accord it significant weight.

Dr. Spagnolo reviewed Mr. P.'s medical records at the Employer's request. He acknowledged that Mr. P.'s x-rays showed consistent evidence of parenchymal abnormalities, consistent with both granulomatous disease and pneumoconiosis. Dr. Spagnolo adopted Dr. Wheeler's conclusion that the x-ray changes on the March 9, 2006 x-ray were most consistent with the effects of a chronic granulomatous process, citing Dr. Wheeler's status as a pre-eminent radiologist in the evaluation of x-rays of persons with occupational exposure and related lung disease. But he did not explain why he favored Dr. Wheeler's conclusions over those of Dr. Alexander and Dr. DePonte, who are also highly qualified board certified radiologists. Additionally, although he included them out in his list of medical records that he reviewed, Dr. Spagnolo did not discuss the significance of the findings on multiple CT scans, which showed fibronodular opacities, pleural thickening, and scarring. Although, as the Employer argues, Dr. Spagnolo had the opportunity to review Mr. P.'s medical records, I find that he did not adequately discuss the significance of those records, nor did he explain why he chose to rely on Dr. Wheeler's interpretation. I do not find his opinion to be entitled to significant weight.

Dr. Repsher also reviewed Mr. P.'s medical records at the Employer's request, and concluded that Mr. P. does not have either medical or legal pneumoconiosis. His statement that there is not radiographic evidence of pneumoconiosis is contrary to the medical evidence of record, and indeed the medical evidence that Dr. Repsher reviewed. Thus, as Dr. Repsher noted, Dr. Rasmussen, Dr. DePonte, and Dr. Alexander all classified Mr. P.'s x-ray as positive for pneumoconiosis. Although Dr. Repsher's report does not make it clear precisely what hospital and treatment records he reviewed, it appears that he did have access to those records, which contain numerous narrative x-ray reports of findings consistent with pneumoconiosis or silicosis. Apparently Dr. Repsher's statement is based on his position that the only "reliable board-certified radiologists and B readers" found no evidence of simple or complicated

pneumoconiosis. But Dr. Repsher did not explain why he thought Dr. Wheeler, Dr. Scott, and Dr. Scatarige are more "reliable" than Dr. Rasmussen, Dr. Alexander, and Dr. DePonte.

Dr. Repsher's report also indicates that, in addition to x-ray reports, he reviewed CT scan reports, which he described as "quite abnormal." But he did not explain why he thought the CT scans were "most consistent" with tuberculosis or sarcoidosis and not pneumoconiosis, or offer any further evaluation of the numerous CT scan reports in the record. I find that Dr. Repsher's report is not well-reasoned or supported by the objective medical evidence of record, and I do not accord it significant weight.

Mr. P.'s records from Johnston Memorial Hospital report his August 2006 admission, where he was diagnosed with numerous problems, including progressive massive fibrosis. Both Dr. Kent and Dr. Cox felt that Mr. P. had cor pulmonale with right ventricular hypertrophy, increased venous pressure, and edema, and severe pulmonary hypertension related to his progressive massive fibrosis.

Similarly, the records from the Holston Valley Medical Center, where Mr. P. was admitted in 2004 and 2001, reflect that he was diagnosed with progressive massive fibrosis and silicosis/pneumoconiosis, as well as hypertension. The treatment records from Pulmonary Associates of Kingsport from 2001 to 2004 document Mr. P.'s treatment for coal workers' pneumoconiosis with progressive massive fibrosis. These records reflect that Mr. P. was seen regularly, and underwent numerous x-rays and CT scans during these years for the evaluation of his respiratory condition.

Considering the totality of the x-ray and CT scan evidence, and placing most reliance on the opinion of Dr. Rasmussen, as supported by Mr. P.'s treatment records, I find that Mr. P. has established by a preponderance of the medical evidence that he has both simple and complicated pneumoconiosis. I also find that the Employer has not offered affirmative evidence that establishes either that the large opacities noted on x-ray by Dr. Rasmussen, Dr. DePonte, and Dr. Alexander are not there, or are due to a process other than pneumoconiosis. Thus, Dr. Wheeler has speculated that the masses he described were "compatible" with conglomerate granulomatous disease, either histoplasmosis or tuberculosis.[4] Dr. Scott felt that the changes he described were "probably" due to tuberculosis, or unknown activity. Dr. Scatarige "favored" tuberculosis as the explanation for the 5 cm. mass he identified on x-ray.

I find that the Employer has offered x-ray and CT scan interpretations that, considered together, do not affirmatively establish that the large opacities are not there, or that they are due to another disease process. The Employer's evidence as a whole suggests the possibility that the process in Mr. P.'s lungs is probably due to something other than pneumoconiosis. But there is no consistent, corroborated, or affirmative evidence that the large opacities identified by Dr. Alexander, Dr. Rasmussen, and Dr. DePonte are not there, or are due to an intervening

[4] Dr. Wheeler also stated, both in his deposition testimony and in his reports, that a biopsy is necessary for a definitive diagnosis, and that this procedure should have been done. I find that this reflects Dr. Wheeler's reliance on a medical or clinical definition of complicated pneumoconiosis, which the Court in *Scarbro* made clear the statute does not incorporate. Thus, it is not necessary for a miner to undergo biopsy, or other pathologic evaluation, to qualify for the irrebuttable presumption under Section 718.304.

pathology. Indeed, the only thing that Dr. Wheeler, Dr. Scatarige, and Dr. Scott can agree on is that the process is probably due to something else, and they offer divergent views about what those possibilities could be.[5] I find that their opinions are not affirmative evidence under *Scarbro*.

Thus, as the other evidence does not affirmatively show that the opacities are not there, or are not what they seem to be, Mr. P.'s x-ray evidence under prong (A) does not lose force, and Section 21(c)(3) and the implementing regulations at 20 C.F.R. § 718.304 compel me to invoke the irrebuttable presumption that Mr. P. is totally disabled due to pneumoconiosis.[6]

CONCLUSION

Based on the totality of the medical evidence, I find that Mr. P. has established that he has pneumoconiosis,[7] and that he is totally disabled due to pneumoconiosis. He is therefore entitled to benefits under the Act.

ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claim of H. P. for benefits under the Act is GRANTED.

IT IS FURTHER ORDERED that the Employer, Premium Energy Inc., shall pay to the Claimant all benefits to which he is entitled under the Act commencing in December 2005.

SO ORDERED.

A

LINDA S. CHAPMAN
Administrative Law Judge

[5] I find that Dr. Wheeler's conclusions are undermined by his assumption that complicated pneumoconiosis is "quite rare," having been seen most frequently in drillers working unprotected during and before World War II. His statements suggest that complicated pneumoconiosis is a thing of the past, but he offered no evidence or support for this assumption.

[6] As the Employer has pointed out, the pulmonary function and arterial blood gas study results do not establish that Mr. P. has a total respiratory disability, nor has any physician stated that Mr. P. does not have the respiratory capacity to return to his previous coal mine work.

[7] As Mr. P. has 27 years of coal mine employment, he is entitled to the regulatory presumption that his pneumoconiosis is due to his coal dust exposure, a presumption that has not been rebutted.

ATTORNEY'S FEES

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).